Isolated (Fiedler's) Myocarditis

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THE following three cases appeared of general interest, as the essential findings at post-mortem were limited to a peculiar form of myocardial degeneration, of which we can find no record in British literature.

CASE I

The patient, D. L., was a female child aged 22 months. On 4/11/43 she had what her mother described as a "choking turn," following which she held her left arm limply and stumbled when attempting to walk. She appeared to be in pain, held herself rigid, with her head retracted, and rolled her eyes from side to side. She did not develop any increase in temperature. On the next day there was twitching of the left side of her face and she refused to use her left arm. Following this her condition appeared to improve.

On examination at this time (15/11/43) the deep reflexes were present. There was a positive Babinski on the left side. The pupils were equal and reacted normally, and the fundi were normal. No lesions were noted in the throat, heart, or lungs. There was some weakness of the left hand.

On 13/11/43 she developed swelling of her face. Swelling of the feet and ankles was seen on 17/11/43 and continued until death.

On 26/11/43 the urine contained sugar, but no albumen. The next day it showed albumen, but no sugar. The œdema gradually increased and there was some increase in the rate of respiration. Respiratory distress, cyanosis, and rales at the base of each lung led to a diagnosis of cardiac decompensation of unknown origin.

Apart from the fact that she was given an immunising course of combined A.P.T. and whooping cough vaccine on 23rd and 30th September, 6th October, and 3rd November, 1943, there was no previous history of any illness. The child was in good social circumstances, fed well, and appeared healthy until the sudden episode on 4/11/43. Death occurred on 6/12/43.

Post-mortem, A. 3910 (Dr. J. E. Morison).—The following essential findings are abstracted from the post-mortem report.

The body is that of a very well-developed, well-cared-for female infant. There is a slight icteric tinge of the conjunctiva. Œdema is present in both ankles and over the sacrum. There is no enlargement of the superficial lymph nodes. On incision, there is considerable œdema of the anterior abdominal wall.

Body Cavities.—The pleural sacs contain about 3 oz. of clear fluid. The

pericardial sac contains 2 oz., whilst the peritoneal cavity is distended with over 1½ pints. There are no adhesions.

Heart.—This is large, relative to the size of the child. The epicardium is smooth and translucent. The right auricle is dilated. The tricuspid valve is thin and delicate. There is no hypertrophy of the right ventricle, but the myocardium is pale. The left auricle, mitral, and aortic valves appear normal. The ventricular muscle has a rather peculiar appearance. It has a pale pink appearance, which has a waxy translucency. Though obviously altered, the muscle has a firm, rigid texture. The endocardium shows a small ante-mortem thrombus in the left ventricle.

Lungs.—The pleura is smooth. There is no enlargement of the lymph nodes at the hilum, and the large bronchi are normal. There is an area of consolidation at the lower posterior part of both lungs. These areas stand out above the level of the adjacent pleura. On section, the alveolar tissue is œdematous, but the areas of consolidation are seen to be almost black in colour from effused blood with loss of alveolar pattern. They are sharply demarcated from the adjacent lung tissues and appear to be infarcts.

Liver.—The liver is enlarged. On section, it presents a strongly contrasting pattern of congested areas alternating with pale yellow areas.

Spleen.—This is about twice the normal size. It is firm and the cut edge remains sharp. The pulp is a dark-red colour and the essential structure is poorly seen. Apart from a marked degree of retro-peritoneal œdema, no other lesions were seen in the thoracic or abdominal viscera.

Brain.—The meninges appear normal. The organ feels soft, especially over the right cerebral hemisphere. There is no tentorial herniation, but the vessels over the right hemisphere are somewhat congested. On section, the convolutions in the region of the right Rolandic fissure show softening. The normal tissue is replaced by a yellowish soft tissue with cystic spaces. No other lesions were seen.

Microscopical Examination.—Sections of all organs were examined. The lungs, brain, and pancreas show no lesions, apart from those due to congestive heart failure.

Heart.—This presents a very varied and unusual picture. Focal areas of recent necrosis are seen. The capillary bed in such foci is generally dilated, but there is no inflammatory infiltration. Adjacent muscle bundles are vacuolated and their fibrillar arrangement indistinct, so that they acquire a rather homogeneous structure. This vacuolisation does not appear to be due to fatty change, for the specific stains show very little stainable fat. It, therefore, probably represents some form of hydropic change.

In other areas the necrotic muscle has disappeared, leaving the stroma collapsed. The nuclei of some of these disintegrated muscle fibres appear to survive. There is a slight proliferation of fibroblasts, but generally the fibroblastic reaction to the muscular lesions is surprisingly slight.

There is little inflammatory infiltration. A few lymphocytic aggregations are seen in the epicardial fat, but, in general, the myocardium is free from infiltration.

The coronary arteries and their branches in the numerous sections examined are

free from disease. The capillary walls in the necrotic areas show swelling of their endothelium, but this appears to be the result, rather than the cause, of the necrosis.

Lungs.—The alveoli show much œdema and scattered aggregates of large mononuclears containing hæmosiderin. There is much distension of the alveolar capillaries. Several sections show necrosis of the alveolar walls, with extravasation of red cells and fibrin into the alveoli. A branch of the pulmonary artery contains an organising thrombus.

Pancreas.—There is œdema of the interstitial tissue and an infiltration of the stroma with lymphocytes. This infiltration is much more intense than that seen in the heart. The acinar and islet tissue cells show no lesions, and the ducts are patent.

Brain.—The area of softening is found to be infiltrated by cerebral histiocytes, many of which are filled with phagocytosed fat. There is no inflammatory infiltration and the glial cells show only early signs of proliferation.

Anatomical Diagnosis.—Isolated myocarditis (Fiedler); intraventricular thrombi; infarcts lungs and brain; subacute venous congestion of lungs, liver, spleen, kidneys, and adrenals; lymphocytic infiltration of pancreas.

Case II

The patient, a farm labourer, aged 16 years, was admitted to the Royal Victoria Hospital on 18/5/43 as a case of nephritis. He gave a history of having suffered from scaret fever in 1935, but there were no known complications. He had been subject to frontal headaches since childhood. He had been fit for his work on the farm and complained of no symptoms until three weeks before his admission, when he began to suffer from shortness of breath. However, he continued at work until 9/5/43, when, following a wetting, his face, legs, and ankles became swollen and he complained of pain in his ankles and calves.

Examination showed him to be a well-nourished youth, with no anæmia, but slight cyanosis and puffiness of the face. His legs and ankles were œdematous. His heart was enlarged, the apex beat diffuse, and in the fifth space half an inch outside the nipple line. The pulse was 160-180. Blood pressure was 115/90 and a mitral systolic murmur was present. The lungs showed dulness at the right base. There was free fluid in the abdomen and the liver appeared enlarged. The urine was scanty and showed albumen + + but no casts. A catheter specimen was sterile. Blood urea 18 mg. per 100 cc. The electro-cardiogram showed a 2—1 auricular flutter, and X-ray confirmed the presence of cardiac enlargement.

His general condition improved under digitalis treatment and there was some diminution of the œdema. The pulse rate, however, remained high. The degree of albuminuria varied and was absent for a few days. The urinary output increased from 12 to 15 oz. per day to 40 to 70 oz.

As the tachycardia and auricular flutter continued, he was given a course of quinidine, starting with gr. V on 20/6/43 and increasing by 5 gr. daily up to 20 gr. per day. On 26/6/43 his pulse rate fell to 32 and his general condition became worse. He felt uncomfortable and was very dyspnœic. Electro-cardiographic examination showed heart block with right ventricular extrasystoles. The quinidine

was stopped and digoxin given. His pulse rate mounted again to 160 and he continued in much the same condition for the subsequent two months. On 18/8/43 he complained of precordial pain and a pericardial friction rub was found. He occasionally showed a slight evening rise of temperature. Blood cultures were sterile on three occasions. The urine continued sterile. Leucocytes were 9,500 per c.mm. Signs of congestive heart failure gradually became prominent. The œdema became generalised and he died on 27/10/43, having been semi-comatose for three days. Some degree of terminal jaundice was noted.

Post-mortem, A. 3856.—Only the essential findings are abstracted.

Body Cavities.—The pericardial sac contains 4 oz. of amber clear fluid; the peritoneal cavity 2 pints. Both pleural cavities are normal.

Heart.—This is enlarged—weight 19½ oz. It is globular in shape and flabby in consistency. All four chambers are dilated. The tricuspid and mitral valves are rendered insufficient by dilatation of the valve rings. The pulmonary and aortic valves are normal. The myocardium is uniformly pale. At the apex of the right ventricular cavity and extending for one inch up the septum is a firm ante-mortem thrombus. Beginning about one inch below the mitral valve ring a thrombus is firmly attached to the left ventricular wall. This extends down to the apex and along the septum to within two inches of the aortic valves. It varies in consistency, but parts of it appear to be organised. The major vessels are patent.

Lungs.—The pleura is smooth. The lungs feel heavier than normal, and, on section, œdema fluid pours from the cut surface. There are several areas of infarction. The bronchi and major pulmonary vessels appear normal.

Liver.—Weighs 3¼ lb. The gall-bladder is thin-walled and contains no stones. The cystic and common bile ducts are patent. On section, the lobules are distinct, and show dark-red central zones with relative pallor of their periphery, resulting in the granular nutmeg pattern of chronic venous congestion. There is slight bile-staining.

Kidneys.—Weigh 5 oz. each. The capsule strips readily, except over a small area in each kidney, where it is adherent to a bright yellow area in the cortex. The surface is smooth, and, on section, the cortical striæ are regular and the cortex of normal width. The medulla and pelvis show no lesions. Section of the yellow areas noted on the surface shows the presence of wedge-shaped infartcs.

Spleen.—Weighs 6 oz. The capsule is smooth. The organ feels firm and the cut surface maintains a sharp margin. At the upper pole there is a yellow area of infarction.

All the other organs appeared normal.

Microscopical Examination—Heart.—The mitral valve and valve ring show no lesions. Scattered through the left ventricle, but more particularly in the subendocardial zone and in the papillary muscles, are irregular areas of fibrous replacement of muscle. These areas of fibrosis vary in their cellularity, the majority being relatively acellular. Others, however, show the presence of many fibroblasts and the formation of new capillaries. More rarely a small mass of necrotic muscle is seen with a commencing fibroblastic reaction. In relation to these areas are small

foci of lymphocytic infiltration, but these are never numerous. A few such focal aggregates are also to be seen in the interstitial tissue unrelated to necrotic muscle or areas of fibrosis. Similar lesions are found in the right ventricular muscle and septum. Overlying the endocardium is an organising thrombus. The degree of organisation varies from field to field and seems to represent different age periods in the formation of the clot. There is no histological evidence of recent or healed rheumatism. The coronary arteries and their branches are normal. The muscle fibres which persist show little change. There is no fatty infiltration. Around the areas of scarring, scattered nuclei appear hypertrophied and an occasional muscle cell, with two large nuclei, is seen. The heart, therefore, shows lesions of at least three different age periods, a sparse lymphocytic infiltration, and a normal coronary system.

Histological examination of the other viscera merely confirms the presence of chronic venous congestion and infarcts in lung, spleen, and kidney. There is no evidence of nephritis.

Anatomical Diagnosis.—Isolated myocarditis (Fiedler); ventricular mural thrombi; chronic venous congestion; infarcts of lung, spleen, and kidney; terminal cardiac jaundice.

CASE III

Admitted 1/4/45. Died 5/4/45.

The patient was a male, aged 48 years. He came to hospital complaining of shortness of breath over the preceding three weeks. For about one year he had noted palpitation on mild exertion and an increasing tendency to the development of fatigue. Before the present illness patient had felt quite well, and had not suffered from any illness of note.

On admission, the patient was seen to be pallid and moderately cyanosed. There was no clubbing of his fingers and no œdema. His pulse was 100, regular, and of poor volume and tension. Blood pressure was 105/85, rising later to 128/105. The apex beat was in the fifth space, 4½ inches from the midline. The heart sounds were faint, with a well-marked gallop rhythm. No murmurs were heard.

Crepitations were present at the lung base. The liver was slightly enlarged. The urine contained albumin. E.C.G. showed a left bundle branch block.

Throughout his stay in hospital there was little change in the patient's condition. Dyspnœa continued, and on 4/4/45 there was hæmoptysis. Enlargement of the liver became more marked. The pulse became weaker and the patient died on the morning of 5/4/45.

Post-mortem, A. 4521 (only the essential findings are abstracted).

Heart.—This is enlarged. Weight 20 oz. It is globular in shape. The epicardium is smooth and there is no excess of fluid in the pericardial sac. The right auricle appears normal. The tricuspid valve ring is dilated, but the valvular cusps are quite thin and show no vegetations. Between the valve cusps and the ventricular wall is a laminated thrombus, extending almost completely round the valve ring. The ventricular muscle appears slightly thickened. The left auricle shows no thrombi, but its wall is thickened and it preserves its shape when cut open. The

mitral valve ring is dilated. The left ventricular endocardium is extensively covered by laminated thrombus. The muscle itself has a peculiar salmon-pink colour, but there is no gross evidence of an infarct. The coronary arteries are patent.

Lungs.—The pleura is smooth. There is no pleural effusion. The lungs feel heavier than normal. The major vessels and bronchi are patent. On section, the alveoli are seen to contain a rather hæmorrhagic œdema fluid. There are no infarcts.

Liver.—Weight 3¾ lb. The gall-bladder is thin-walled and the cystic and common bile ducts are patent. On section, the lobular pattern is accentuated, due to the congestion of each central zone and the relative pallor of the peripheral zone. The radicles of the bile-ducts and portal veins are patent.

Spleen.—Weight 7 oz. The capsule is smooth and the organ feels firm. On section, the pulp is dark-red from congestion. The malpighian corpuscles are rather indistinct.

All other organs appear normal.

Microscopical Examination—Heart.—Numerous sections from all cavities were examined. The endocardium of both ventricles is seen to be covered by blood clot. Organisation is progressing, and is, in general, most advanced within the lumina of the thebesian veins. Over some areas the clot is completely organised. The underlying endocardium shows fibrous thickening and focal collections of cells, which are mainly lymphocytes, are present. Similar lymphocytic infiltrations, but of lesser intensity, are seen in the fibrous septa of the myocardium. Individual muscle fibres, more especially in the subendocardial layer, appear necrotic, whilst there are small scattered areas of fibrosis unrelated to blood vessels. The coronary arteries are patent. The intima is a little thickened, but nowhere is any atheromatous lesion seen of sufficient extent to be held responsible for the myocardial fibrosis.

Other organs show only the results of congestive heart failure. Unlike the other two cases, there are no infarcts.

Discussion

Clinically, these cases resemble each other in showing a congestive heart failure with intermittent albuminuria and embolic episodes. There was nothing in their history or symptoms, however, to point to any possible ætiological factor which might have allowed of the more exact diagnosis of the condition. Pathologically, the lesions appear to be identical, though the longer duration of illness in cases II and III has allowed of the development of more fibrosis in the areas of myocardial degeneration. The curious fact emerges from the pathological examination that, apart from the presence of infarcts, there is no disease process in any other viscus or tissue which might be regarded as being associated with the myocardial lesion. Such "isolated" (Sellentin, 1904) myocardial lesions were first described by Steffen in 1888, but the name of Fiedler, who reported a similar process in 1890, is most often associated with the condition. Saphir (1941) stated that "one is justified in accepting the occurrence of isolated myocarditis in the sense of a more or less diffuse inflammatory lesion if every known cause for this type of myocarditis is ruled out, and if the myocarditis is found in the absence of any major pathological condition involving either the endocardium and pericardium or the entire body."

The present three cases seem, therefore, to justify the diagnosis of "isolated myocarditis."

Scott and Saphir in 1929 were able to collect thirty-six cases, and a cursory review of the literature shows that at least forty-one additional cases have been reported since that date. The disease, though rare, has therefore probably been often overlooked and is not so uncommon as the paucity of reports before Scott and Saphir's paper would seem to indicate.

Examination of the cases reported in the literature as examples of isolated or Fiedler's myocarditis indicates the probability that several conditions of different ætiology have been included under this term. Some authors stress the infiltration of the interstitial tissues of the heart by inflammatory cells. This infiltration may or may not be associated with necrosis of the myocardial fibres. Others (Jonas, 1939; Magner, 1939; Miller, 1935) describe a granulomatous type of lesion which apparently simulated the granulomata of tuberculosis, syphilis, or sarcoidosis. The lesion found in the present cases, however, appears to be essentially different from either of these types. Inflammatory infiltration is by lymphocytes, and nowhere are these present in large numbers. They tend to occur mainly in relationship to the areas of necrosis or repair and may well be a reaction to the products of the degenerating muscle rather than called forth in response to the presence of any bacterial or virus agency. It would appear, indeed, that the essential lesion is a degeneration of the muscle cell—a degenerative process which is peculiar in that, in some cells at least, it affects the sarcoplasm rather than the nuclei (Case I). Muscle nuclei occasionally survive in the degenerated area, and may proliferate to produce giant forms. The method whereby the degenerating muscle is removed is not easily apparent, for there is little evidence of phagocytosis. This degeneration of the myocardium is evidently associated with alterations in the endocardium, which favour the initiation of thrombi. In cases I and III there is well-marked endocardial fibrosis, and this is also present in Case II, though here the presence of subendocardial scarring suggests that the organising thrombi may be related to foci of necrotic muscle.

It would seem, then, that the cases reported as Fiedler's myocarditis are really heterogeneous in type and that no purpose is served in endeavouring to find for them a common ætiology. Cases showing a very similar pathological process have been reported by Gouley, McMillen, and Bellett (1937). These authors record four cases of cardiac failure occurring in pregnancy which showed foci of muscle degeneration, with a tendency to preservation of the muscle nuclei and subsequent fibrosis. Cases have been described in infants by Lindberg (1938), Mazzeo (1929), Maslow and Ledderer (1933), Singer (1932), Smith and Stephens (1938), and Bluhdorn (1924) and others. Under the title of "myocarditis perniciosa," Boikan (1931) described a similar case in a woman of 28 years. This author attempted to formulate a classification of isolated myocarditis, but beyond the recognition of acute and chronic and recurrent forms his classification adds little to our knowledge. In his report, Lindberg (1938) suggests that the lesion may represent the end stage of the beri-beri heart, and more recently this opinion has been supported by Smith

and Furth (1943) and Toreson (1944). However, there is little evidence that chronic beri-beri heart has produced a lesion similar to that under discussion, and Levy (1930) found vitamin B of no therapeutic value in similar cases. Remissions occurred without any vitamin therapy in the patients reported by Smith and Furth (1943). Whilst we have no detailed knowledge of the diet of Case I, the child was living in excellent social conditions and at autopsy appeared well nourished. Case II, the farm labourer, was on regular hospital diet for several months before death and showed both healed and acute lesions at post-mortem. Case III was of the artisan class, was not addicted to alcohol, and had the same rations as the rest of the community. Furthermore, the recent experimental work of Pantridge (1948) in this department has shown that in the pig thiamin deficiency results in necrosis and degenerative changes in the conducting system of the heart. Apart from some foci in the auricles, necrosis of myocardial muscle was not a feature of the deficiency. It is unlikely, therefore, that the myocardial degeneration was due to an avitaminosis. The occurrence of similar lesions in the heart in association with apparently allergic skin reactions has also suggested an allergic mechanism for the cardiac injury. Maxwell and Barrett (1934) report a case developing after a severe dermatitis, due to applications of a sulphur ointment. Brown and McNamara (1940) found a similar process in the heart following the administration of arsphenamine, whilst Nelson's case (1934) was associated with an exfoliative dermatitis. Against this interpretation must be urged the fact that in experimental sensitisation the cardiac lesions are much more akin to those seen in rheumatic carditis and polyarteritis nodosa, and the lesions so produced tend to involve the interstitial septa rather than the muscle tissue of the heart (McKeown, 1947).

The histological examination in these three cases does suggest a degenerative, rather than an inflammatory, lesion. The absence of any important incidents in the clinical histories of the patients renders it impossible to state what may have been the cause of this degeneration.

Summary

1. Three cases of heart failure in the absence of any of the usual cardiac lesions are reported.
2. In all three were areas of myocardial degeneration, with various stages of scar formation and fibrosis of the endocardium.
3. Mural thrombi were present in the ventricles and had produced infarcts.

My thanks are due to Drs. Boyd Campbell and F. M. B. Allen for the clinical notes, and to Drs. J. E. Morison and Y. MacIlwaine for post-mortem reports on two of the cases. I am indebted to Mr. D. Mehaffey, A.R.P.S., for the illustrations.

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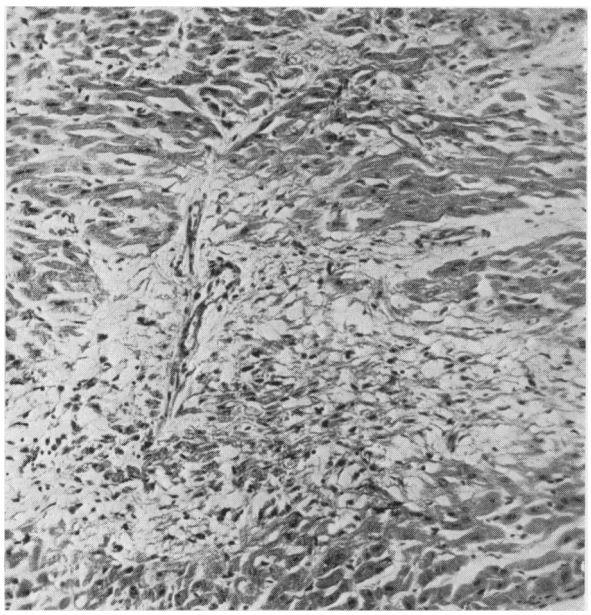

Fig. 1

Case I. A.3910. To show one of the recent focal areas of muscle necrosis. Adjacent fibres show vacuolisation, and there is little inflammatory infiltration.

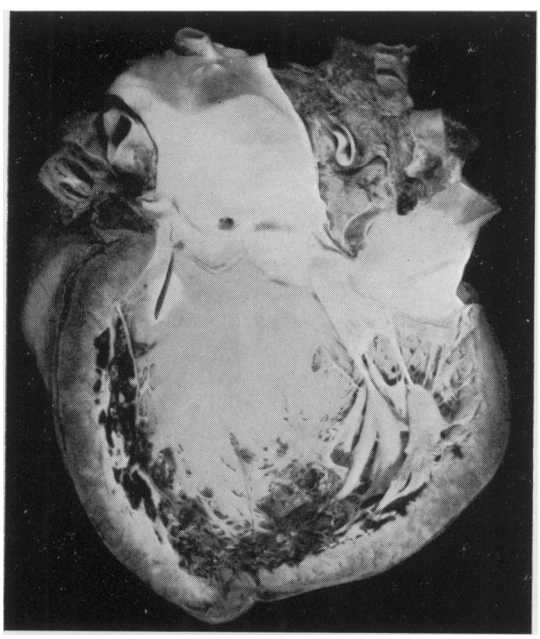

Fig. 2

Case II. A.3856. To show the dilated heart with its lining of ante-mortem thrombus.

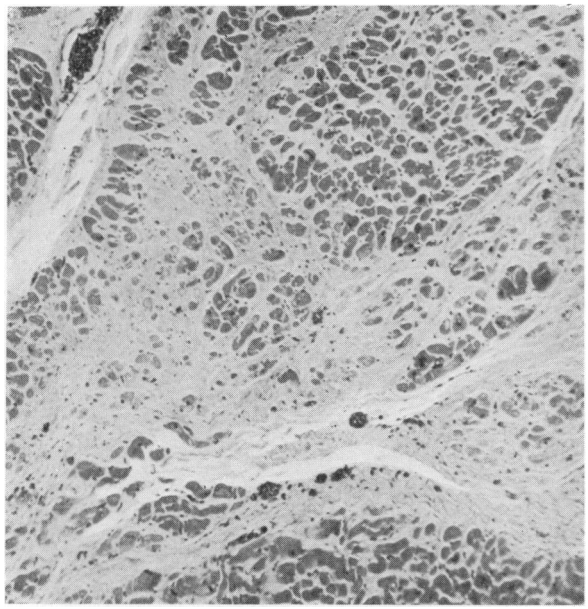

Fig. 3

Case II. To show the irregular fibrosis of the myocardium. There is no cellular infiltration.

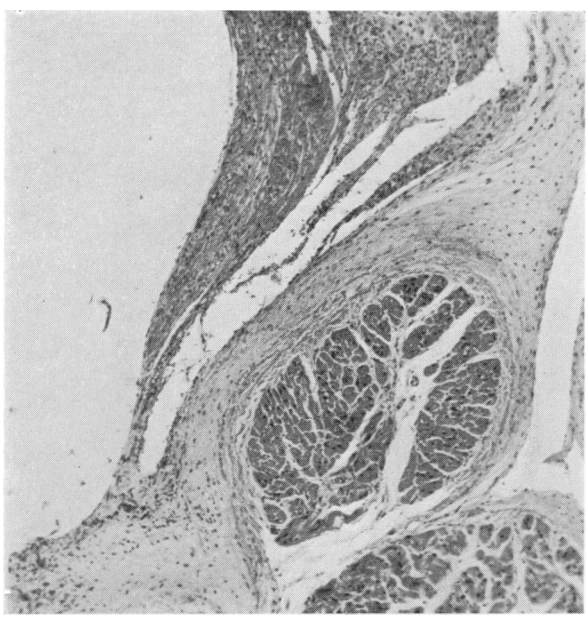

Fig. 4

Case II. To show the fibrous thickening of the endocardium resulting from organisation of mural thrombus.

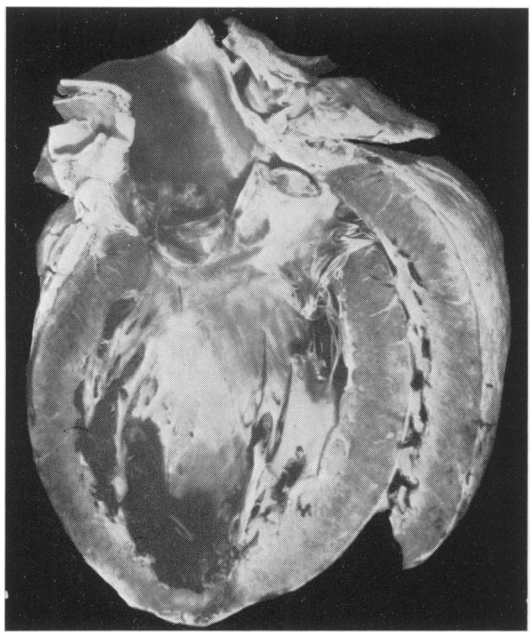

Fig. 5

Case III. A.4521. To show the dilated heart and the extensive mural thrombus.

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REVIEWS

BEDSIDE DIAGNOSIS. By Charles Mackay Seward, M.D., F.R.C.P.(Edin.), Edinburgh: E. & S. Livingstone Ltd. 17s. 6d. net.

DR. SEWARD'S aim is to provide a "diagnostic approach" in the elucidation of fundamental presentations of disease. By the systematic analysis of some twenty significant symptoms and signs, he shows that a diagnosis can usually be made at the bedside; his scheme is logical and orderly and the special tests judiciously chosen. Due weight and sympathetic consideration are given to psychogenic symptoms, and this, together with the author's essentially practical viewpoint, make the book a useful contribution to clinical teaching. Dr. Seward's treatment of his subject emphasises the integration required in clinical appraisal and diagnosis; the student who reads this book will cease trying to "match" the patient with cut-and-dried text-book descriptions of diseases, and will thus shorten the long road to professional maturity.

While "Bedside Diagnosis" was not intended as a comprehensive collection of all major symptoms and signs, the reviewer feels that its value would be enhanced still further by the inclusion of chapters on "Arthritis" and "Coma." R. G. V.

GROW UP—AND LIVE. By Eustace Chesser. Pp. 295. Pelican Books. 1s. 6d.

DR. EUSTACE CHESSER is well known for his work on sex education and marriage guidance, and this Pelican book, written for adolescents, will maintain his high reputation.

The book begins with a brief survey of the processes of evolution and development, and continues through the description of the functions of body and mind, to the attainment of maturity and an integrated personality. Sex education receives adequate treatment, due attention being paid both to the physical and the psychological aspects.

The author writes in simple, easily understood language, but does not make the mistake of talking down to his audience. His methods are neither dogmatic nor didactic, but consist of suggestions and guidance, from which the adolescent must choose his own line of conduct. The emphasis is on self-discipline, accompanied by tolerance, and consideration for others, and the ethical aspects are duly stressed.

Although this book is primarily for adolescents, it should prove very useful to youth leaders—and to parents. M. E. L.